(FILE 'HOME' ENTERED AT 10:57:26 ON 21 JUN 2001)

FILE 'BIOSIS' ENTERED AT 10:57:34 ON 21 JUN 2001

L1 42 S OOCYTE? AND (NO DONOR OR NITRIC OXIDE DONOR OR NOS OR NOS
IND
L2 159342 S FERTIL?
L3 12 S L1 AND L2
L4 14 S OOCYTE? AND (NOS OR NITRIC OXIDE SYNTASE) AND INHIBIT?
L5 4 S BOVINE AND OOCYTE AND (NITRIC OXIDE)
L6 6 S BOVINE AND OOCYTE? AND (NITRIC OXIDE)
L7 26 S HUMAN AND OOCYTE? AND (NITRIC OXIDE)

FILE 'CAPLUS' ENTERED AT 11:35:31 ON 21 JUN 2001

L8 19 S HUMAN AND OOCYTE? AND (NITRIC OXIDE)

FILE 'MEDLINE' ENTERED AT 11:40:51 ON 21 JUN 2001

L9 26 S HUMAN AND OOCYTE? AND (NITRIC OXIDE)

L4 ANSWER 5 OF 14 BIOSIS COPYRIGHT 2001 BIOSIS

AB This study was conducted to determine whether endothelial-derived nitric oxide synthase (eNOS) affects meiotic maturation of mouse **oocytes** in vitro. Cumulus-**oocyte** complexes (COC) were isolated from ovarian follicles of 27-day-old PMSG-primed wildtype (WT), and eNOS-knockout (eNOS-KO) females, and cultured in drops of medium under oil at 37degreeC for 16-18 hr. Experiment 1 was carried out to determine effects of eNOS deficiency on the ability of COC to mature in vitro. To determine whether acute synthesis of nitric oxide (NO) was required for **oocyte** maturation, COC collected from WT mice were cultured in medium without (control) or with different doses of Nomega-nitro-L-arginine methyl ester (L-NAME), an **inhibitor** of **NOS** (exp. 2). To assess effects of NO deficiency on the kinetics of germinal vesicle breakdown (GVBD), COC from WT and eNOS-KO females were observed for 3.5 hr. COC from WT females were also incubated in medium without or with L-NAME (exp. 3 and 4). After the culture period, cumulus cells were removed, and **oocytes** were counted and classified as metaphase II (M II), metaphase I (M I) or showing atypical (degenerative) morphology. To determine viability and nuclear morphology of **oocytes**, they were stained with fluorescein diacetate or 4,6-diamidine-2'-phenylindole dihydrochloride, respectively. There were no differences in body weights but ovarian weights were lower in eNOS-KO mice compared with WT mice ($P < 0.05$). Ovaries from eNOS-KO mice contained fewer COC collected relative to WT mice ($P < 0.01$). Maturation of COC from eNOS-KO mice or WT **oocytes** treated with L-NAME resulted in a lower percentage of **oocytes** at M II stage ($P < 0.01$ and $P < 0.05$, respectively) and a higher percentage of **oocytes** at M I or atypical stages compared with those from WT ($P < 0.01$ and $P < 0.05$, respectively). Many **oocytes** that showed either an arrest in M I stage or abnormal morphology were not viable. Several **oocytes** in M II stage demonstrated abnormalities in distribution of maternal chromosomes. Our data demonstrate that eNOS-derived NO is a key modulator of **oocyte** meiotic maturation in vitro. These results support our previous observations in vivo and indicate that eNOS/NO has independent functions in both **oocyte** maturation and follicular/**oocyte** development.

ACCESSION NUMBER: 2000:167668 BIOSIS
DOCUMENT NUMBER: PREV200000167668
TITLE: Nitric oxide is essential for optimal meiotic maturation of murine cumulus-**oocyte** complexes in vitro.
AUTHOR(S): Jablonka-Shariff, Albina; Olson, Lisa M. (1)
CORPORATE SOURCE: (1) NCP-U4B, Monsanto Company, 800 N. Lindbergh Blvd., Saint Louis, MO, 63167 USA
SOURCE: Molecular Reproduction and Development., (April, 2000) Vol. 55, No. 4, pp. 412-421.
ISSN: 1040-452X.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English

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L1 ANSWER 5 OF 42 BIOSIS COPYRIGHT 2001 BIOSIS

AB We investigated the extent to which NO participates in the developmental competence (**oocyte** maturation, fertilization and embryo development to blastocyst) using an in vitro culture system adding sodium nitroprusside (SNP), **NO donor**, and **NOS** inhibitor (N-omega-nitro-L-arginine methyl ester, L-NAME). We also assessed the effects of NO/**NOS** system on blastocyst implantation using an in vitro trophoblast outgrowth assay. The treatment of low concentrations of SNP (10-7 M) significantly stimulated meiotic maturation to metaphase II stages in cumulus enclosed **oocytes**, In contrast, 10-3 and 10-5 M L-NAME demonstrated a significant suppression in resumption of meiosis. This inhibition was reversed by the addition of SNP. No development beyond the four-cell stage was observed by the addition of high concentration of SNP (10-3 M). Inhibition of embryo development, especially the conversion of morulae to blastocysts, was also observed in the treatment of lower doses of SNP (10-5 and 10-7 M). Similarly, inhibition of NO by **NOS** inhibitor resulted in the dose-dependent inhibition of embryo development and hatching rates, but the concomitant addition of SNP with L-NAME reversed the inhibitory effect by each SNP or L-NAME treatment. Furthermore, low concentration of SNP (10-7 M) but not high concentration of SNP (10-3 M) significantly stimulated trophoblast outgrowth, whereas the addition of L-NAME suppressed the spreading of blastocysts in a dose-dependent manner. These results suggest that NO may have crucial roles in **oocyte** maturation and embryogenesis including the process of implantation. The observed differences in required amount of NO and the sensitivity to cytotoxicity of NO in each developmental stage embryos may also suggest that NO/**NOS** system is tightly regulated in developmental stage specific manner.

ACCESSION NUMBER: 2001:129039 BIOSIS
DOCUMENT NUMBER: PREV200100129039
TITLE: Requirement of nitric oxide for murine **oocyte** maturation, embryo development, and trophoblast outgrowth in vitro.
AUTHOR(S): Sengoku, Kazuo (1); Takuma, Naoyuki; Horikawa, Michiharu; Tsuchiya, Keiko; Komori, Harumi; Sharifa, Dinara; Tamate, Kenichi; Ishikawa, Mutsuo
CORPORATE SOURCE: (1) Department of Obstetrics and Gynecology, Asahikawa Medical College, Midorigaokahigashi 2-1, Asahikawa, 0788510: ksen@asahikawa-med.ac.jp Japan
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